



# UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE  
United States Patent and Trademark Office  
Address: COMMISSIONER FOR PATENTS  
P.O. Box 1450  
Alexandria, Virginia 22313-1450  
www.uspto.gov

|  |             |                      |                         |                  |
|--|-------------|----------------------|-------------------------|------------------|
| APPLICATION NO.  | FILING DATE | FIRST NAMED INVENTOR | ATTORNEY DOCKET NO.     | CONFIRMATION NO. |
| 10/037,341   | 01/04/2002  | David Baltimore      | 75723-ZA/JPW/GJG        | 6591             |
| 23432 7590 01/04/2007<br>COOPER & DUNHAM, LLP<br>1185 AVENUE OF THE AMERICAS<br>NEW YORK, NY 10036 |             |                      | EXAMINER<br>GUZO, DAVID |                  |
|  |             |                      | ART UNIT                | PAPER NUMBER     |
|  |             |                      | 1636                    |                  |
| SHORTENED STATUTORY PERIOD OF RESPONSE   |             | MAIL DATE            | DELIVERY MODE           |                  |
| 3 MONTHS   |             | 01/04/2007           | PAPER                   |                  |

**Please find below and/or attached an Office communication concerning this application or proceeding.**

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

**Office Action Summary**

Application No.

10/037,341

Applicant(s)

BALTIMORE ET AL.

Examiner

David Guzo

Art Unit

1636

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

- 1) ☒ Responsive to communication(s) filed on 02 October 2006.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

- 4) ☒ Claim(s) 1-30 and 32-90 is/are pending in the application.
- 4a) Of the above claim(s) 1-30, 32-65 and 74-90 is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 66-73 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

**Application Papers**

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☒ The drawing(s) filed on 04 January 2002 is/are: a) ☒ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

**Priority under 35 U.S.C. § 119**

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some \* c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
  2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
  3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

\* See the attached detailed Office action for a list of the certified copies not received.

**Attachment(s)**

- |   |   |
|---|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892)   | 4) <input type="checkbox"/> Interview Summary (PTO-413)<br>Paper No(s)/Mail Date. _____ |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948)  | 5) <input type="checkbox"/> Notice of Informal Patent Application                       |
| 3) <input checked="" type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08)<br>Paper No(s)/Mail Date <u>11/26/02; 1/24/06; 10/2/06</u> . | 6) <input type="checkbox"/> Other: _____  |

**Detailed Action**

**Election/Restriction**

Applicant's election with traverse of Group XXIV, Claims 66-73 and 89-90 in the reply filed on 10/2/06 is acknowledged. The traversal is on the ground(s) that the examiner has not made the case for the different inventions being independent and distinct because all groups relate to NF- $\kappa$ B. Additionally, applicants assert that there would not be a undue burden on the examiner if all of the inventions were examined together because a search of the prior art relevant to NF- $\kappa$ B would necessarily uncover any art relevant to each of the purported Groups. This is not found persuasive because the examiner has shown the different groups are independent or distinct for the reasons outlined in the previous Office Action. For example, the different Groups recite nucleic acids vs. proteins, methods of regulating expression of genes in cells regulated by binding of NF- $\kappa$ B to enhancer elements vs. controlling HIV expression in cells, vs. agonists or antagonists of nuclear proteins which bind to transcriptional regulatory elements of immunoglobulin genes, etc. Each Group would require a separate search even though they all involve, either directly or peripherally, NF- $\kappa$ B or some activity of NF- $\kappa$ B. For example, a search of a method for controlling HIV expression in cells (Group XXI) would not be co-extensive with a search of polyclonal or monoclonal antibodies (Group XIII) or a search of a method for detecting binding of cellular nuclear protein to DNA, etc. A different field of search would be required to identify art on each of the different inventions. For example, searching for antibodies reactive to NF- $\kappa$ B

Art Unit: 1636

would not unearth art on a method for controlling HIV gene expression in cells or phage OCT-2 or a NF- $\kappa$ B – I $\kappa$ B protein complex, etc. As noted in MPEP 808.02:

Where the inventions as claimed are shown to be **independent or distinct** under the criteria of MPEP § 806.05(c) - § 806.06, the examiner, in order to establish reasons for insisting upon restriction, must explain why there would be a serious burden on the examiner if restriction is not required. Thus the examiner must show by appropriate explanation one of the following:

(A) Separate classification thereof: This shows that each invention has attained recognition in the art as a separate subject for inventive effort, and also a separate field of search. Patents need not be cited to show separate classification.

(B) A separate status in the art when they are classifiable together: Even though they are classified together, each invention can be shown to have formed a separate subject for inventive effort when the examiner can show a recognition of separate inventive effort by inventors. Separate status in the art may be shown by citing patents which are evidence of such separate status, and also of a separate field of search.

(C) A different field of search: Where it is necessary to search for one of the inventions in a manner that is not likely to result in finding art pertinent to the other invention(s) (e.g., searching different classes /subclasses or electronic resources, or employing different search queries, a different field of search is shown, even though the two are classified together. The indicated different field of search must in fact be pertinent to the type of subject matter covered by the claims. Patents need not be cited to show different fields of search.

Since the inventions of the different groups are independent or distinct and a burdensome search would be required to search all of the different inventions, restriction is proper.

With regard to newly added claims 89-90, said claims fall within a separate, patentably distinct group and are directed to subject matter distinct and/or independent from the subject matter of elected Group XXIV. Newly added claims 89-90 read on a method for reducing expression in a human cell of a gene, the expression of which has been induced by an external influence that activates NF- $\kappa$ B to act as an intracellular messenger to transmit a signal that induces expression of the gene from the plasma

membrane of the cell to the nucleus of the cell, which method comprises within the cell inhibiting transmission of the signal so as to thereby reduce expression of the gene in the cell. These claims read on inhibiting a signal(s) subsequent to activation of a gene by NF-kB while the claims of the elected invention read on methods of regulating NF-kB mediated gene activity in a cell by altering NF-kB activity in the cell, not by inhibiting signal(s) transmitted after normal NF-kB activation of the gene in question. The invention of Group XXIV and the invention described in claims 89-90 read on regulating gene expression by acting on two different aspects of NF-kB mediated gene expression and a search of one would not be co-extensive with a search of the other and hence would be burdensome.

The requirement is still deemed proper and is therefore made FINAL.

Claims 1-30, 32-65 and 74-90 are withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to a nonelected invention, there being no allowable generic or linking claim. Applicant timely traversed the restriction (election) requirement in the reply filed on 10/2/06.

### **Priority**

Priority for the claimed subject matter in claims 66-68 and 70 is granted back to the filing date of the 07/341,436 ('436) application (4/21/1989). Priority for the subject matter of claim 69 (method of enhancing NF-kB) is granted back to the filing date of the

06/946,365 ('365) application (12/24/1986). Priority for the subject matter of claims 71-73 is granted back to the filing date of the 07/791,898 ('898) application (11/13/1991).

No application filed prior to the 07/341,436 application discloses a method of regulating (which reads on increasing **or inhibiting**) NF-kB mediated gene expression in a cell, comprising altering NF-kB activity in the cell. Applications prior to the '436 application recite methods of inducing or increasing NF-kB activity in cells but do not recite methods of inhibiting or decreasing NF-kB activity in cells. The subject matter of claim 69 is first disclosed in the '365 application, the prior application does not disclose the claimed method of enhancing NF-kB activity in **any cell** (as is currently claimed). With regard to claims 71-73, applications prior to the '898 application do not provide an enabling disclosure (or provide a written description) of expression systems comprising a binding site for NF-kB operably linked to a promoter and gene of interest and culturing the cells under conditions for expressing any gene in any cell. With regard to the binding site consensus sequence recited in claim 72 and the list of sequences comprising said consensus sequence recited in claim 73, these limitations were first disclosed in the '436 application.

### **35 USC 102 Rejections**

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 66-68 and 70 are rejected under 35 U.S.C. 102(b) as being anticipated by the Physician's Desk Reference (PDR: 1985) pages 1811-13; Griffith I (Griffith et al., Ann. Surg. 196 (9/82): 324-329) or Griffith II (Griffith et al., J. Thorac. Cardiovasc. Surg. 99 (12/84): 952-957) as evidenced by Holschermann et al., Circulation 96 (12/97) 4232-4238.

Applicants claim a method of regulating NF- $\kappa$ B-mediated gene expression in a cell, comprising altering NF-KB activity in the cell and a method of regulating transduction in a cell of an extracellular signal by NF- $\kappa$ B, comprising altering NF- $\kappa$ B activity in the cell, wherein NF- $\kappa$ B activity is reduced. Applicants also claim a method of regulating NF- $\kappa$ B-mediated expression of a selected gene in a cell, comprising introducing into the cell a substance which regulates NF- $\kappa$ B activity in the cell.

PDR (1985), Griffith I and, Griffith II teach administration of cyclosporin A (CsA) to (into) cells in cardiac patients, which is shown from the teaching of Holschermann, to inherently regulate (reduce) NF- $\kappa$ B activity and thus would inhibit expression of genes whose transcription is regulated by NF- $\kappa$ B activity or regulate transduction in the cell of an extracellular signal by NF- $\kappa$ B since NF- $\kappa$ B activity is altered (reduced) as an effect of administration of CsA. The inhibition is done by reducing binding of NF- $\kappa$ B to NF- $\kappa$ B recognition sites, which also decreases the level of NF- $\kappa$ B not bound in a NF- $\kappa$ B - I $\kappa$ B complex, inhibiting the passage of NF- $\kappa$ B into the nucleus of cells, inhibiting modification of an I $\kappa$ B protein, and inhibiting degradation of an I $\kappa$ B protein.

Specifically, the PDR 1985 reference teaches that CsA should be administered before and after surgery for 1-2 weeks at a dose of about 15 mg/kg/d, followed by a

decrease of 5% per week to a final level of 5-10 mg/kg/day (see p. 1813). When monitoring whole blood levels, a 24-hour trough value of 250-800 ng/ml CsA appeared to minimize side effects and rejection effects. Griffith I reports the administration of 5-10 mg/kg/d of CsA (average 8 mg/kg/d) (See Abstract); while Griffith II reports the administration of 2-30 mg/kg/d (average 7.5--8 mg/kg/d) (See Abstract) to obtain a targeted blood level of CsA of about 1000ng/ml.

Holschermann provides extrinsic evidence that the PDR 1985, Griffith I, and Griffith II references inherently anticipate the subject claims. Holschermann essentially repeated the tests disclosed in the Griffith I and II references by administering  $3.4 \pm 0.3$  mg/kg/day CsA to cardiac transplant patients, resulting in blood levels of  $681 \pm 176$  ng/ml (See p. 4233). PBM cells were isolated from the blood of the patients before and after CsA therapy, and nuclear extracts from the cells were prepared. Holschermann then conducted an EMSA assay using nuclear extracts (see Figure 4) which is the same assay format taught by applicants for determining whether compounds (i) reduce NF- $\kappa$ B activity and (ii) reduce binding of NF- $\kappa$ B to NF- $\kappa$ B recognition sites. Holschermann confirms that administering CsA to cardiac patients as taught by the prior art PDR 1985 and Griffith I and II references necessarily inherently reduces NF- $\kappa$ B activity (and binding of NF- $\kappa$ B to NF- $\kappa$ B recognition sites). In cells obtained from transplant recipients during low baseline CsA blood levels (before CsA administration), strong NF- $\kappa$ B binding activity was detected (Fig. 4), whereas cells separated from blood in the presence of high CsA concentrations exhibited decisively reduced NF-KB binding activity. Specificity of the binding reaction was shown by the competition with unlabeled



consensus oligonucleotides (See p. 4236). Holschermann also showed that the administration of CsA to these patients as taught in the prior art PDR 1985 and Griffith I and II references reduced Tissue Factor (TF) gene transcription, which is recognized as being regulated by NF- $\kappa$ B: "Indeed, the marked activation of the NF- $\kappa$ B transcription factor, which is known to play a major role in the regulation of the TF gene, was prevented in the presence of high CsA blood concentrations." Id. at 4237. Thus, Cyclosporin A, as administered in the prior art PDR 1985 and Griffith I and II references:

- a. regulated (inhibited) expression of a gene whose transcription is regulated by NF- $\kappa$ B
- and; b. regulated (diminished) transduction in a cell of an extracellular signal mediated by NF- $\kappa$ B. See MPEP 2131.01 (evidence of inherency).

Claims 66-67, 69 and 70 are rejected under 35 U.S.C. 102(b) as being anticipated by Gescher et al. or Hunter et al.

Applicants claim a method of regulating NF- $\kappa$ B-mediated gene expression in a cell, comprising altering NF-KB activity in the cell and a method of regulating transduction in a cell of an extracellular signal by NF- $\kappa$ B, comprising altering NF- $\kappa$ B activity in the cell, wherein NF- $\kappa$ B activity is enhanced. Applicants also claim a method of regulating NF- $\kappa$ B-mediated expression of a selected gene in a cell, comprising introducing into the cell a substance which regulates NF- $\kappa$ B activity in the cell.

It is noted that administration of phorbol esters such as 12-O-tetradecanoyl-phorbol-13-acetate (TPA) to human cells (such as A549 and A431 cells) inherently induces (enhances) NF- $\kappa$ B activation (See for example, Kang et al., Cell. Mol. Life Sci.,

Art Unit: 1636

2005, Vol. 62, pp. 1146-1155 and Jang et al., Biochem. Biophys. Res. Comm., 2005, Vol. 328, pp. 70-77). Kang et al. teaches that TPA augments the promoter activity of the AR gene via the activation of NF- $\kappa$ B and protein kinase while Jang et al. teaches that TPA induced TLR2 gene expression via NF- $\kappa$ B activation.

Gescher et al. (Cancer Research, September 1985, Vol. 45, pp. 4315-4321, see whole article, particularly the Abstract and pp. 4316-4317) teaches administration of TPA ( $10^{-8}$  M) to human A549 cells, wherein said administration inherently enhances NF- $\kappa$ B activity and Hunter et al. (Nature, 1984, Vol. 311, pp. 480-483, see whole article, particularly the Abstract and Fig. 1) teaches administration of TPA (100 ng/ml) to human A431 cells, wherein said administration inherently enhances NF- $\kappa$ B activity and therefore the expression of genes induced by binding of NF- $\kappa$ B (i.e. TLR2 and AR). Gescher et al. and Hunter et al. therefore teach the claimed invention.

Claims 71-72 are rejected under 35 U.S.C. 102(b) as being anticipated by Pasleau et al.

Applicants claim a method of positively regulating NF- $\kappa$ B-mediated gene expression in a cell, comprising: a) introducing into the cell a gene construct comprising a gene of interest, a DNA sequence which is the binding site of NF- $\kappa$ B and a promoter for the gene; and b) maintaining the cell under conditions appropriate for expression of the gene. The binding site is represented by the following consensus sequence:

      C      C  
GGGRATYYAC or equivalents thereof.  
      T      T

It is noted that the CMV IE promoter/enhancer has several NF- $\kappa$ B binding sites and is subject to regulation by NF- $\kappa$ B (See, for example, Lee et al., Eur. J. Biochem., 2004, Vol. 271, pp. 1094-1105, see especially Fig. 1). The NF- $\kappa$ B binding site consensus sequence is represented in the CMV promoter/enhancer.

Pasleau et al. (Gene, June 1985, Vol. 38, pp. 227-232, see whole article, particularly the Abstract, Fig. 1 and p. 230) recites introducing into the cell a gene construct comprising a gene of interest (bovine growth hormone gene, bGH), a DNA sequence which is the binding site of NF- $\kappa$ B and a promoter for the gene (CMV IE promoter/enhancer) and maintaining the cell under conditions appropriate for expression of bGH. Pasleau et al. therefore teaches the claimed invention.

Claims 71-73 are rejected under 35 U.S.C. 102(b) as being anticipated by Cullen.

Applicants claim a method of positively regulating NF- $\kappa$ B-mediated gene expression in a cell, comprising: a) introducing into the cell a gene construct comprising a gene of interest, a DNA sequence which is the binding site of NF- $\kappa$ B and a promoter for the gene; and b) maintaining the cell under conditions appropriate for expression of the gene. The binding site is represented by the following consensus sequence:

          C      C  
GGGRATYYAC or equivalents thereof and wherein the consensus sequence is present  
          T      T  
in the HIV LTR.

Cullen (Cell, Sept. 1986, Vol. 46, pp. 973-982, see whole article, particularly the Abstract; "Results" section on pp. 973-974; Tables 1-2) recites introducing into a cell a gene construct comprising a gene of interest (human interleukin-2, IL-2), a DNA

sequence which is the binding site of NF- $\kappa$ B and a promoter for the gene (HIV LTR) and maintaining the cell under conditions appropriate for expression of IL-2. Cullen therefore teaches the claimed invention.

Claims 71-72 are rejected under 35 U.S.C. 102(b) as being anticipated by Banerji et al. (Cell, 1981, Vol. 27, pp. 299-308) or Humphries et al. (Cell, 1982, 1982, Vol. 30, pp. 173-183).

Applicants' invention is as described above.

Humphries et al. (See whole article, particularly the Abstract ; Fig. 1; pp. 179-180) and Banerji et al. (See whole article, particularly the Abstract; Figs. 1 and 4; pp. 302-304) both recite introducing into a cell a gene construct comprising a gene of interest ( $\beta$ -globin or  $\alpha$ -globin or  $\delta$ -globin genes), a DNA sequence which is the binding site of NF- $\kappa$ B (in the SV40 enhancer) and a promoter for the gene and maintaining the cell under conditions appropriate for expression of IL-2. Cullen therefore teaches the claimed invention. Applicants indicate in the instant specification (see for example, pages 74, 83 and Table 2) that the SV40 enhancer has NF- $\kappa$ B binding site(s). Banerji et al. and Humphries et al. therefore teach the claimed invention.

### **Statutory Double Patenting**

A rejection based on double patenting of the "same invention" type finds its support in the language of 35 U.S.C. 101 which states that "whoever invents or discovers any new and useful process ... may obtain a patent therefor ..." (Emphasis added). Thus, the term "same invention," in this context, means an invention drawn to identical subject matter. See *Miller v. Eagle Mfg. Co.*, 151 U.S. 186 (1894); *In re*

Art Unit: 1636

*Ockert*, 245 F.2d 467, 114 USPQ 330 (CCPA 1957); and *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970).

A statutory type (35 U.S.C. 101) double patenting rejection can be overcome by canceling or amending the conflicting claims so they are no longer coextensive in scope. The filing of a terminal disclaimer cannot overcome a double patenting rejection based upon 35 U.S.C. 101.

Claims 66-73 are provisionally rejected under 35 U.S.C. 101 as claiming the same invention as that of claims 66-73 of copending Application No. 10/037,415. This is a provisional double patenting rejection since the conflicting claims have not in fact been patented.

### **Obviousness Type Double Patenting Rejections**

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Claims 66-68 and 70 are rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1-5, 9-17, 20-63, 88-176 and 192-203 of U.S. Patent No. 6,410,516 (hereafter the '516 patent). Although the conflicting claims are not identical, they are not patentably distinct from each other because both sets of claims read on methods of regulating NF- $\kappa$ B mediated gene expression in a cell, comprising altering (inhibiting) NF- $\kappa$ B activity in the cell. The instant claims are generic to the claims recited in the '516 patent. That is, the recited claims of the '516 patent fall entirely within the scope of the instant claims, or in other words, the instant claims are anticipated by the claims of the '516 patent. For example, the various methods for inhibiting expression of NF- $\kappa$ B mediated gene expression in cells recited in the '516 patent are encompassed within the instant broad methodologies (i.e. the instant methods encompass any method of regulating NF- $\kappa$ B mediated gene expression in any cell).

No Claims are allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to David Guzo, Ph.D., whose telephone number is (571) 272-0767. The examiner can normally be reached on Monday-Thursday from 8:00 AM to 5:30 PM. The examiner can also be reached on alternate Fridays.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Irem Yucel, Ph.D., can be reached on (571) 272-0781. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only.

Art Unit: 1636

For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

David Guzo  
December 19, 2006

  
DAVID GUZO  
PRIMARY EXAMINER